It’s a Fit-up!
The Case of the ‘Rheumatoid Arthritis’

Victim: Cartilage cells and bone

Profession:
Bone is the highly mineralised tissue forming the skeleton. It is strong but relatively inflexible. Where bones meet to form joints, some form of cushioning is required to allow joint articulation and absorb the forces resulting from movement of the body. Cartilage (formed from cells called chondrocytes) sheaths the opposing ends of bones forming joints – cushioning and protecting them.

Location:
The tissue affected is found within the synovial capsule (the structure that encloses and protects joints in the human body).

Details of the case:
The underlying cause has yet to be clearly identified and may actually be due to a variety of factors – it is almost certainly a form of autoimmunity. The progress of the disease is well characterised – large numbers of neutrophils (phagocytes) infiltrate the synovial membrane lining the synovial capsule. This initiates changes in the membrane, causing it to thicken and alter its structure. This draws the attention of other elements of the immune system. Macrophages, T cells, and B cells are all associated with the further progression of the disease – macrophages in particular produce important inflammatory factors. Perhaps due to the somewhat isolated nature of the synovial capsule it is easy for a self-sustaining immune reaction to become established, exacerbating the disease progression.

The consequences:
The thickened synovial membrane (known as ‘pannus’) can directly erode cartilage and bone, aided by neutrophils releasing factors that directly damage the cartilage surface. Signalling molecules known as cytokines can directly induce chondrocytes, and other cells called fibroblasts, to produce enzymes that break down the cellular structure within the joint. Unfortunately once joint damage has occurred it is irreversible – and the self-sustaining nature of the condition is problematical in this respect.

How can Immunology help?
Without yet having a clear understanding of the underlying causes, we aren’t yet able to stop the disease at point of origin. However, one of the major successes in combating the condition came from the realisation that an inflammatory factor called ‘tumour necrosis factor alpha’ (TNFα) played an important role in escalating the immune response. By directly neutralising this molecule using an antibody specific to it, major success in relieving symptoms has been achieved. Cases unresponsive to this therapy may be treated with currently experimental techniques, such as chemotherapy or even a form of stem cell transplantation, whereby the cells making up a patient’s immune system are replaced – getting rid of the malfunctioning cells. Such approaches are drastic however – the more we understand the underlying causes, the more chance of an effective preventative approach to the disease.

Can you help? Immunology needs you!

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